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The effect of age and structural lesions on postictal language impairment

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Summary The duration of postictal language dysfunction following a temporal lobe complex partial seizure (TLCPS) is longer when the seizure originates in the dominant hemisphere. However, the effects of older age and the presence of a structural lesion ipsilateral to the area of origin of the seizure remain unknown. Postictal language delay (PILD) was analyzed in relation to age and presence of a structural lesion in 47 patients, 28 with dominant TLCPSs and 19 with nondominant TLCPSs (total 173 seizures). Mean ages of the groups were 32.5 years (range: 16—68) and 36.1 years (range: 21—50), respectively. Nonsclerotic structural lesions were found by magnetic resonance imaging in 13 patients, eight with seizures in the dominant hemisphere and five with seizures in the nondominant hemisphere. Age did not affect PILD regardless of the lateralization of the seizures. The presence of a structural lesion significantly prolonged PILD only in the patients with nondominant TLCPS ($p = 0.019$). In conclusion, the anatomical site of seizure onset may not be the only determinant of the nature of the postictal state. PILD can provide important information on seizure localization and spread.

Introduction

Seizures are commonly followed by transient neurological symptoms and signs. In 1855, Todd[1] described the transient postictal paralysis that now bears his name. Engel[2] defined postictal phenomena as ''manifestations of seizure-induced reversible alterations in neuronal function, but not structure''. Some of the factors that have been suggested to prolong the postictal state are structural lesions, prolonged seizures, and older age.[3—6] The study of postictal language delay (PILD) in humans has been limited by the lack of a consistent, quantitative method to document characteristics and duration. To overcome this obstacle,

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we developed a technique for measuring postictal language delay during video-electroencephalogram (EEG) monitoring. The measurements were found to be reliable, making it possible for clinicians to accurately localize temporal lobe complex partial seizures (TLCPSs) and to better identify patients with right or bilateral language dominance.[7,8]

The aim of the present study was to assess effect of age and the presence of a structural lesion on the PILD. Older patients may also have atypical manifestations of complex partial seizures.[9] We hypothesized that older patients have more structural lesions (e.g., strokes) and therefore, prolonged PILD.

Methods

Since 1988, a standardized language test has been administered during the ictal and postictal states to all patients undergoing video-EEG monitoring at the Epilepsy Monitoring Unit of University Hospital, University of Cincinnati, Ohio. As soon as a seizure is detected clinically, a trained observer (nurse, EEG technician) asks the patient to start reading a printed test phrase aloud (''They heard him speak on the radio last night''), and then to keep repeating the phrase until his/her reading is judged to be correct. This period is defined as the postictal language delay (PILD). Patients were included in the study if they met the following criteria: (1) surgery for temporal lobe seizures between 1988 and 1996 resulting in more than 90% seizure reduction during at least 1 year of postoperative follow-up; (2) adequate postictal language assessment (i.e., language testing instituted within 60 s after the electrographic termination of the seizure) during complex partial seizures without secondary generalization; (3) left hemisphere language dominance, as assessed by intracarotid amobarbital test or direct electrical stimulation of the language cortex. Patients with right or bilateral language dominance were excluded. All brain magnetic resonance imaging (MRI) scans were reviewed to assess the presence of structural lesions. The histopathological report was reviewed when biopsy or resective surgery had been performed. The presence of hippocampal atrophy alone, with or without signal abnormality, was considered nonlesional. Using the GENMOD procedure, we entered the data from each seizure into a model that takes into account possible correlations among multiple observations in individual patients.[10] Specifically, we tested the relationship of age (as a continuous variable) and the presence of a structural lesion with PILD in dominant and nondominant TLCPS's.

Table 1 Lesion etiologies in relation to lateralization.

Dominant temporal lobe seizures	
Angioma	2
Arteriovenous malformation	1
Hamartoma	1
Low-grade glioma	2
Post surgical encephalomalacia	2
Total	8
Nondominant temporal lobe seizures	
Arteriovenous malformation	1
Angioma	1
Hamartoma	1
Low-grade glioma	1
Traumatic encephalomalacia	1
Total	5

Results

Forty-seven patients with a total of 173 seizures were eligible for the study, 28 (19 men) with dominant TLCPSs and 19 (10 men) with nondominant TLCPSs. Mean age of the groups was 32.5 years (range: 16—68) and 36.1 years (range: 21—50), respectively, and duration of epilepsy was 4—36 and 4—38 years, respectively. Thirteen patients had structural lesions, eight in the dominant hemisphere and five in the nondominant hemisphere (Table 1). There was no relationship between PILD and age regardless of the side of the epileptiform discharge or the presence of a structural lesion ($p = 0.4$, dominant-TLCPS group; $p = 0.5$, nondominant-TLCPS group).

There was no statistically significant association between age and the presence of a structural lesion for either dominant or nondominant TLCPSs.

In the nondominant-TLCPS group, the PILD was significantly longer in the patients who had a structural lesion than in those who did not ($p = 0.019$; Table 2). In the dominant-TLCPS group, the presence of a lesion was associated with a shorter PILD, but values did not reach statistical significance ($p = 0.076$; Table 2). The number of seizures for each patient and range of PILD are summarized in Tables 3 and 4.

Discussion

Well-known postictal neurological phenomena include amnesia, Todd's paralysis, flat affect and cortical blindness,[11] hemianopsia,[12] language dysfunction,[13] bulimia,[14] and pulmonary edema.[15] In the present study, we examined postictal language impairment, which is readily quantified and has